GIS

Sheet no. 1

Pathology

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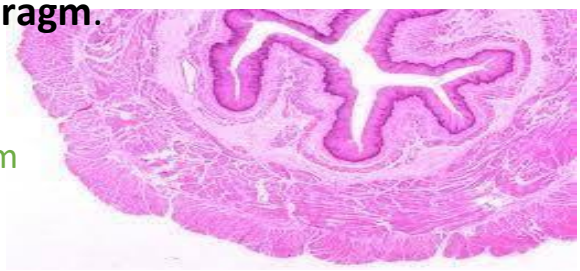
Diseases of the esophagus

Esophagus: is a hollow, highly distensible muscular tube, with central lumen that extends from the epiglottis to the gastroesophageal junction GEJ (or we call it gastroesophageal sphincter(physiological sphincter) that separate the esophagus from the stomach) , located just above the diaphragm.



In histologic picture: we can see that the esophagus

is lined by stratified non-keratinized squamous epithelium

then we can see the submucosa then muscular layer

Note: esophagus non- peritonized: (doesn't have serosa from outside like other part of GIT).

Diseases of the esophagus:

- **1. Obstruction: mechanical or functional.**
- **2. vascular diseases: varices. (دوالي المريء)**
- **3. Inflammation: esophagitis.**
- **4. Tumors.**

Start with mechanical Obstruction: disease that can be seen with naked eyes
These conditions may be **congenital or acquired.**

Examples:

1- Atresia (will be explained below)

2- Fistulas (opening between two hollow organ)

3- Duplications (condition of double-lumen esophagus)

4- Agenesis (v rare) (esophagus is absent)

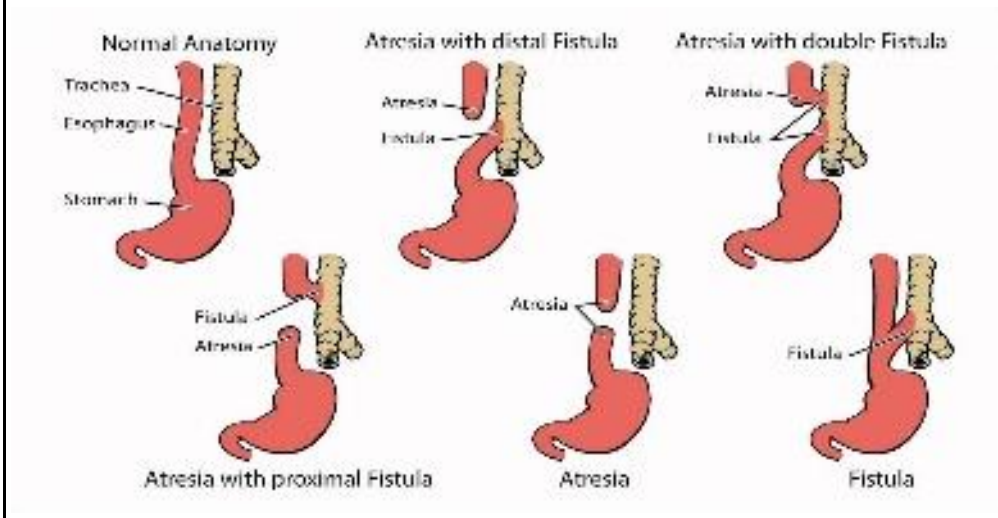
5- Stenosis (narrowing of the esophagus lumen)

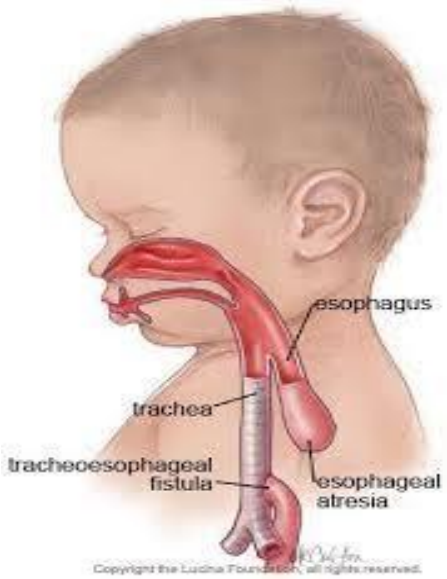
► Atresia:

- A condition in which thin, usually short segment of esophagus that is **noncanalized cord replaces a segment of esophagus**, so the formation of a continuous esophageal tube is interrupted. therefore, whatever passes through the esophagus would result in an obstruction, it's a congenital disease (usually the baby born with atresia and has difficulty in feeding).

-Most common location: at or near the tracheal bifurcation

- Usually associated with a fistula (connection between two hollow spaces) **connecting the upper or lower esophageal pouches to a bronchus or to the trachea.**





Note :The tracheoesophageal fistula (TEF) fistula can lead to breathing problems (aspiration pneumonia) if saliva from the mouth or stomach contents enter the trachea and lungs.

Note : Neonates with TEF or esophageal atresia are unable to feed properly. Once diagnosed, prompt surgery is required to allow the food intake and to prevent the previously-mentioned complications

Clinical presentation:

Clinical presentation: Shortly after birth: regurgitation during feeding (milk cannot pass and start to regurgitate) **and it Needs to prompt surgical correction (rejoin).**

Complications if it with fistula: Aspiration (breathing foreign objects into airways, usually food, saliva or stomach contents), Suffocation, Pneumonia, Severe fluid and electrolyte imbalances (sever dehydration)

►**Esophageal stenosis:** (narrowing of the esophagus lumen)

- **Most of the times acquired and not congenital (Acquired>>>Congenital)**
- **Fibrous thickening of the submucosa & atrophy of the muscularis propria will cause narrowing of the lumen generally**
- **Due to previous inflammation and scarring**

Explanation: when stomach acid and other irritants damage the lining of the esophagus over time. This leads to inflammation and scar tissue, which causes the esophagus to narrow.

Causes:

1. Chronic GERD.

2. Irradiation (cancer patients treated with radiotherapy are prone to esophageal stenosis).

3. Ingestion of caustic agents (chemicals, alkaline or acidic material).

Clinical presentation:

-Dysphagia (difficulty in swallowing) is usually the main symptom with stenosis.
-Difficulty eating solids typically occurs long before problems with liquids

Functional obstruction: (related to abnormality in the innervation affecting the motility of the esophagus)

-Efficient delivery of food and fluids to the stomach requires coordinated waves of peristaltic contractions. Impaired peristaltic movements →no propelling of food →Functional obstruction

-Esophageal dysmotility: dis-coordinated peristalsis or spasm of the muscularis

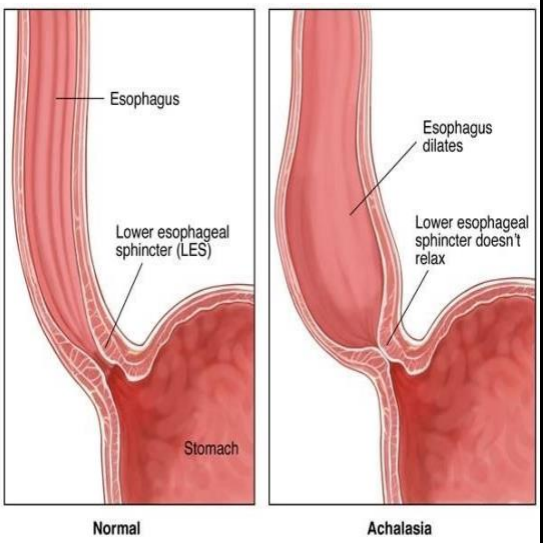
Achalasia is the most important cause of functional obstruction.

Achalasia is characterized by a triad:

1-Incomplete Lower esophageal sphincter (LES) relaxation (when food reaches the LES, the sphincter must relax to permit the passage of food to the stomach, in achalasia the sphincter would take longer duration to relax)

2-Increased Lower esophageal sphincter (LES) tone (in the resting state muscle will be spastic , due to increased tone of the sphincter is detected)

3-Esophageal aperistalsis (absent of peristaltic movement)

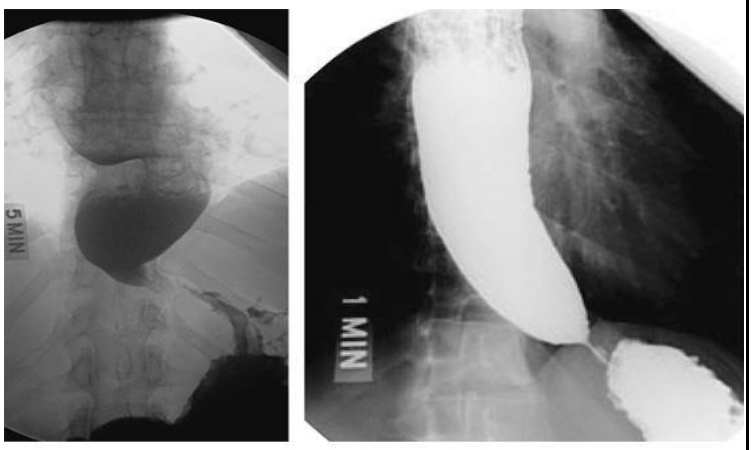


Food enter the esophagus → accumulate in the esophagus → dilatation of the esophagus.

Primary causes are more common than secondary causes . Primary >>>secondary.

Diagnosis:

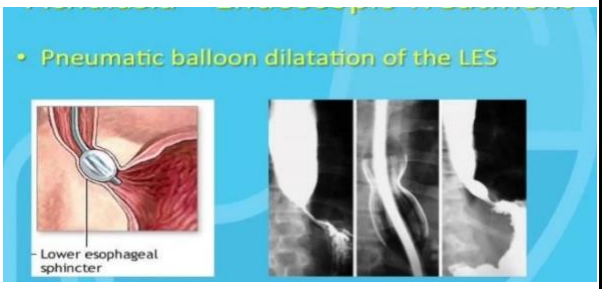
We use **barium swallow** Barium Sulfate is a metallic compound that shows up on X-ray and is used to help see abnormalities in the esophagus and stomach. When taking the test, the patient drinks a preparation containing this solution. The X-rays track its path through the digestive system



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

Treatment:

Treatment options essentially focus on relaxing the LES



Endoscopic balloon dilatation of LES (pneumatic dilation -some patients may have to undergo repeated dilation treatment in order to achieve symptom improvement) OR suigical correction

Primary achalasia (most common) :

-Caused by failure of distal esophageal inhibitory neurons and (the problem is intrinsic of inhibitory neurons in distal esophagus)

-(Idiopathic) Primary mean unknown cause I can't identify the secondary cause for the problem

Secondary achalasia:

-Degenerative changes in neural innervation, either intrinsic to the esophagus or within the extraesophageal vagus nerve or the dorsal motor nucleus of the vagus

-One of the causes of secondary achalasia is Chagas disease: its an infection by Trypanosoma Cruzi (parasite) causes destruction of the myenteric plexus → failure of LES relaxation → esophageal dilatation

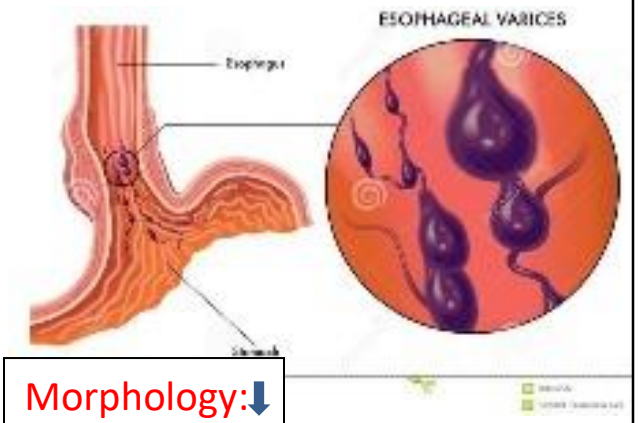
Clinical presentation :

-Difficulty in swallowing (dysphagia)

-Regurgitation or aspiration of food of respiratory tract complicated by pneumonia

-Sometimes chest pain.

Vascular diseases: Esophageal Varices



-Tortuous dilated veins within the submucosa of the distal esophagus and proximal stomach. Any scratch can cause sever bleeding

Usually located in distal esophagus and proximal stomach (around gastroesophageal junction)

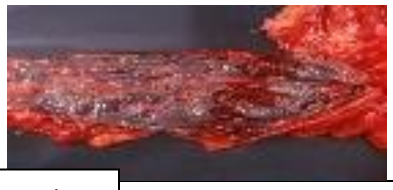
It caused by portal-hypertension (when blood shut to gastroesophageal junction and cause Esophageal Varices)

-Diagnosis by:

endoscopy or angiography.

Morphology:⬇

This picture from autopsy study we can see the blue discoloration its dilated submucosal vein



grossly

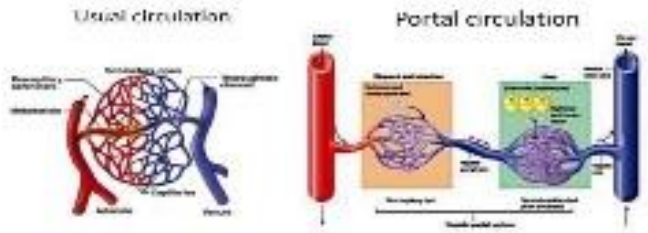
We don't do biopsy for histologic section we can take after autopsy we can see dilated submucosal vein under squamous mucosa

microscopely

Pathogenesis :

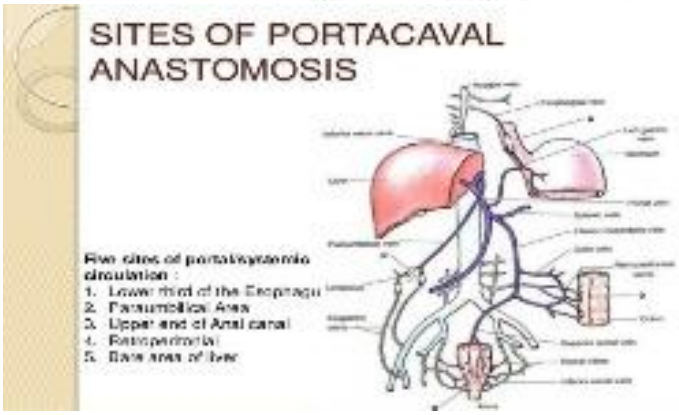
In normal scenario

Portal system

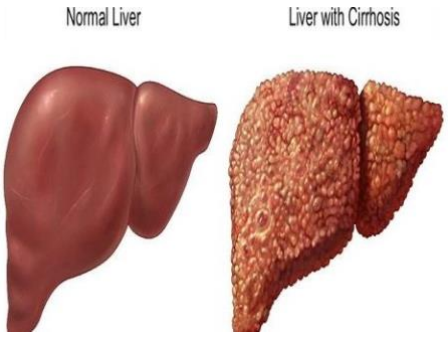


Portal circulation :blood from GIT go to portal vein the to the liver (detoxification) then to inferior vena cava

but when disease like cirrhosis impede portal blood flow this will cause portal hypertension and shunt of blood take place in esophageal and cause esophageal varices



Potal hypertension → collateral channels in distal esophagus → shunt of blood from portal to systemic circulation → dilated collaterals in distal esophagus → varices



- Distal esophagus : site of Porto-systemic anastomosis

Causes of portal hypertension : Cirrhosis is most common Alcoholic liver disease Hepatic schistosomiasis 2nd most common worldwide

Clinical Features of esophageal varices :

we have mild and sever varices depend on degree of dilatation **mild form are asymptomatic** this patients should go under regular endoscopy and try to teat it before dilated vein enlarge and carry's high risk of bleeding , **Rupture leads to massive hematemesis and death , 50% of patients die from the first bleed despite interventions , Death due to: hemorrhage, hepatic come, and hypovolemic shock,** and for people who survive from first bleeding they can **rebleed again (20%).**

Worst case scenario:

Disease that impede portal blood flow → portal hypertension → **collateral channels in distal esophagus ,shunt of blood from portal to systemic circulation** → dilated collaterals in distal esophagus → **varices ,rupture of vessels and severe bleeding** → death due to hypovolemic shock

ESOPHAGITIS:

Inflammation or irritation of the esophagus. Common causes include :

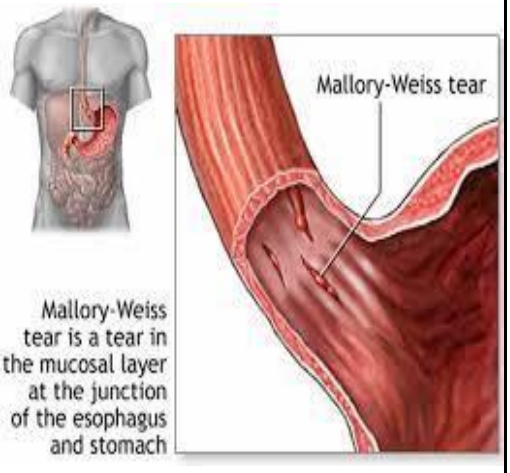
› **Esophageal Lacerations.**(its not an inflammation but most sources add it)

› **Mucosal Injury.** Due to drink alkaline or acid material

› **Infections (viral, fungal, and rarely bacterial).**

› **Reflux Esophagitis.**

› **Eosinophilic Esophagitis.**



Esophageal Lacerations :

Mallory-Weiss tears are most common laceration

-Refers to bleeding from a laceration in the mucosa at the gastroesophageal junction**. This is usually caused by severe/violent vomiting and retching, forceful vomiting** causes tearing of the mucosa (superficial) at the junction

-Present with hematemesis (fresh blood vomiting) , bright red blood

Pathogenesis: **failure of gastroesophageal musculature to relax prior to antiperistaltic contraction associated with vomiting. Gastric contents cause the esophageal wall to stretch and tear**

They are superficial and heal quickly without any surgical intervention

Chemical Esophagitis :

Inflammation induce by chemical substance like ingestion of **Corrosive acids or alkalis** will cause **Damage to esophageal mucosa by irritants** another causes like **Alcohol , Excessively hot fluids, Heavy smoking , Medicinal pills** some times when patient take pills and did not drink water with it very well pill will stuck in esophagus resulting in **pill-induced esophagitis** example of these pills **(doxycycline and bisphosphonates) , Iatrogenic** (disease caused by medical treatment) like : **(Chemotherapy, Radiotherapy , GVHD(graft versus host disease))**

Clinical symptoms & morphology:

The morphologic changes consist of ulceration and acute inflammation

Esophagitis due to chemical injury generally causes only self-limited pain, particularly odynophagia (pain with swallow)

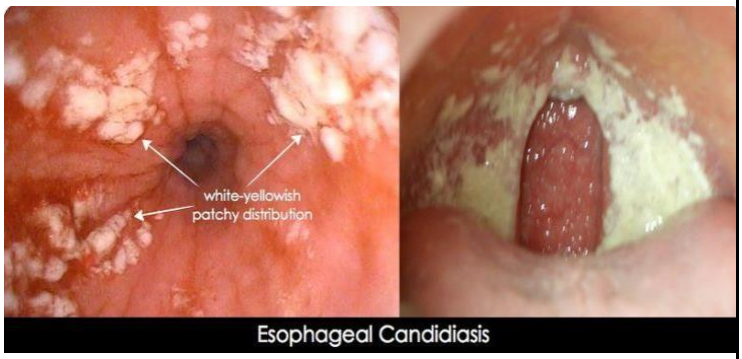
Hemorrhage (may present with hematemesis) , on long run stricture and stenosis due chemical , or perforation may occur in severe cases.

Infectious esophagitis :

Mostly in debilitated or immunosuppressed It is an esophagitis caused by infectious pathogens. It could be viral (HSV , CMV) ,

fungal (candida >>> mucormycosis & aspergillosis) or bacterial 10% Least common

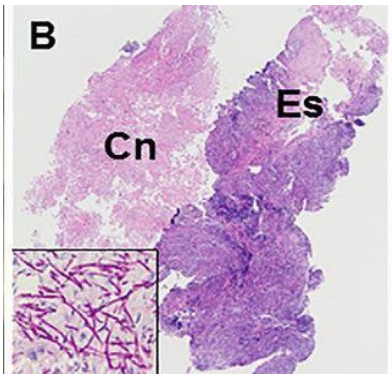
CANDIDIASIS : During endoscopy, you will see in the patient **grayish-white adherent pseudomembranes on the esophagus and Composed of matted fungal hyphae and inflammatory cells**



Esophageal Candidiasis

In the picture you will see oral thrush in the oral cavity. Candida is part of our normal flora, but it does no harm when the patient is immunocompetent

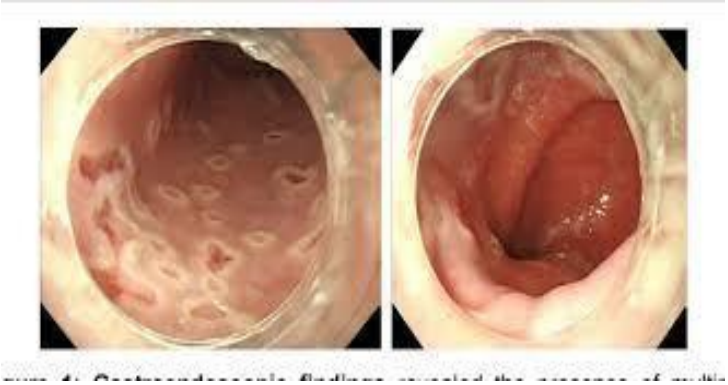
Under the microscope we looking for fungal hyphae



Fungal hyphae →

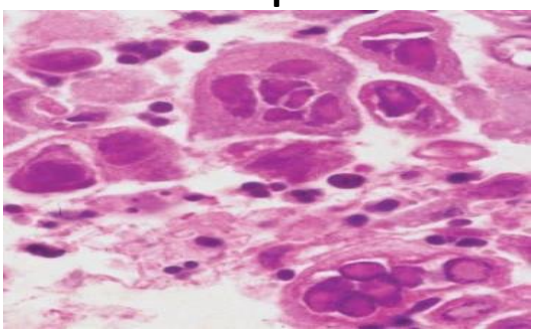
VIRAL ESOPHAGOITIS: the two most important viruses: HSV,CMV

Herpes viruses (HSV):



Presence of punched out (deep, heated-up rounded edges) ulcers.

Histopathologic : there are certain changes called viral cytopathic effect on cells and example of these changes : Nuclear viral inclusions and Degenerating epithelial cells ulcer edge and Multinucleated epithelial cells



in histologic picture: we can see multi-nucleated giant cells (the cell has more than one nucleus) and Within the nucleus inside there is a whiteish spot called an intra-nuclear inclusion

CYTOMEGALOVIRUS (CMV) :

CMV infects the stromal fibroblasts and the endothelial cells lining the capillaries under the mucosa.

Presence of **shallow ulcers** (unlike in HSV they were punched out ulcers).

Biopsy: nuclear and cytoplasmic inclusions in capillary endothelium and stromal cells

Inclusions inside the nucleus

IMPORTANT NOTES:

We can do certain Immunohistochemistry stains to detect HSV or CMV.

If you diagnose a patient with fungal or viral esophagitis, you need to check if he isimmunocompromised (it mostly affects immunocompromised patients).

HSV infect epithelial cells while CMV in addition other cells like endothelial cells .

Good luck <3